Amendments to the Specification:

Please enter the following amendments in regards to the typographical errors found at paragraph [00023]. Namely replace the phrase "FGB-2" with the phrase "FGF-2", and replace the phrase "BFGF" with the phrase "bFGF".

A marked-up replacement paragraph [00023] is shown below:

~~FGB-2~~FGF-2 is a pluripotent mitogen capable of stimulating migration and proliferation of a variety of cell types including fibroblasts, macrophages, smooth muscle and endothelial cells. In addition to these mitogenic properties, FGF-2 can stimulate endothelial production of various proteases, including plasminogen activator and matrix metalloproteinases, induce significant vasodilation through stimulation of nitric oxide release and promote chemotaxis. FGF-2 is present in the normal myocardium and its expression is potentiated by hypoxia or hemodynamic stress. Because of its heparin-binding properties, FGF-2 binds avidly (Kd 10.sup.-9 M) to endothelial cell surface heparin sulfates. This interaction serves to prolong effective tissue half-life of the FGF-2 protein, facilitates its binding to its high-affinity receptors and plays a key role in stimulation of cell proliferation and migration. ~~BFGF~~ bFGF also possesses a plethora of other biological effects such as the ability to stimulate NO release, to synthesize various proteases, including plasminogen activator and matrix metalloproteinases, and to induce chemotaxis. Homozygous deletion of the bFGF gene is associated with decreased vascular smooth muscle contractility, low blood pressure and thrombocytosis. One interesting aspect of bFGF is its biological synergy with VEGF. Thus, a combination of ~~BFGF~~ bFGF and VEGF is far more potent than bFGF alone in inducing angiogenesis in vitro and in vivo. Furthermore, bFGF induces VEGF expression in smooth muscle and endothelial cells.